



THE UNIVERSITY *of* EDINBURGH

Edinburgh Research Explorer

Abnormal sleep in patients with epileptic or dissociative (non-epileptic) seizures: a polysomnography study

Citation for published version:

Popkirov, S, Stone, J & Derry, CP 2018, 'Abnormal sleep in patients with epileptic or dissociative (non-epileptic) seizures: a polysomnography study', *European Journal of Neurology*.
<https://doi.org/10.1111/ene.13798>

Digital Object Identifier (DOI):

[10.1111/ene.13798](https://doi.org/10.1111/ene.13798)

Link:

[Link to publication record in Edinburgh Research Explorer](#)

Document Version:

Peer reviewed version

Published In:

European Journal of Neurology

Publisher Rights Statement:

This is the author's peer-reviewed manuscript as accepted for publication.

General rights

Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

Take down policy

The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact openaccess@ed.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.



Article type : Original Article

Abnormal sleep in patients with epileptic or dissociative (non-epileptic) seizures: a polysomnography study

Stoyan Popkirov^{1*}, Jon Stone², Christopher P. Derry²

¹Department of Neurology, University Hospital Knappschaftskrankenhaus, Ruhr University Bochum, Bochum, Germany

²Centre for Clinical Brain Sciences, Western General Hospital, University of Edinburgh, Edinburgh, United Kingdom (institution where work was performed)

***Corresponding author:**

Dr. Stoyan Popkirov, Klinik für Neurologie, Universitätsklinikum Knappschaftskrankenhaus Bochum, In der Schornau 23-25, 44892 Bochum, Germany. Tel.: +492342993706. Fax: +492342993719. Email: popkirov@gmail.com.

Running title: Sleep and seizures

Keywords: sleep apnea; periodic limb movements; polysomnography; psychogenic nonepileptic seizures; dissociative seizures; epilepsy

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/ene.13798

This article is protected by copyright. All rights reserved.

Disclosure of potential conflicts of interest: The authors report no potential conflicts of interest relevant to this study

Funding: none.

ABSTRACT

Background: The aim of this study was to identify the prevalence of sleep disorders and measure the objective sleep quality in patients with seizure disorders.

Methods: Patients admitted for video electroencephalography monitoring were prospectively recruited and polysomnography was performed on the third night of monitoring.

Results: Four out of 44 (9%) epilepsy patients and 2/22 (9%) patients with dissociative seizures were found to have mild sleep-disordered breathing (SDB). Three (7%) epilepsy patients were found to have mild or moderate obstructive sleep apnea-hypopnea syndrome (OSAHS) and 3 (14%) patients with dissociative seizures had mild or moderate OSAHS. Most patients with SDB or OSAHS were overweight or obese. Time awake after sleep onset (WASO) was high in both groups. There were no significant differences in sleep architecture between the groups except for a difference in average N3 sleep stage proportion. Periodic limb movements (PLM) were common in both groups, and 27% of patients with dissociative seizures had both high PLM rates and high arousal indexes, suggesting a high prevalence of probable PLM disorder in that group (compared to 9% in the epilepsy group).

Conclusions: Our findings contradict the commonly reported high comorbidity of OSAHS and epilepsy, and question its purported clinical relevance. In patients with dissociative seizures high rates of PLM were found. In both patient groups high WASO times were

indicative of sleep disruption, which can have an epileptogenic effect and is known to increase dissociative tendencies.

INTRODUCTION

The phenomenology of seizures, both epileptic and dissociative, has often invited oneiric analogies, such as John Hughlings-Jackson's "dreamy state" or Pierre Janet's "fits of somnambulism".^{1,2} But beyond the common denominator of a change in awareness, there are various clinicopathological connections between seizure disorders and sleep. In people with epilepsy, sleep may be disrupted by nocturnal epileptic activity or anti-epileptic drugs, and, conversely, sleep states can influence the onset and course of seizures.³⁻⁵ Among sleep disorders presumably associated with epilepsy, obstructive sleep apnea-hypopnea syndrome (OSAHS) has received particular attention.⁶ While high rates of comorbidity are often reported,⁷⁻⁹ most studies on this topic have considerable limitations in sample size and methodology.³ In a larger, two-step study on 283 unselected epilepsy patients using structured interviews for initial screening and polysomnography in symptomatic patients, an OSAHS rate of 10% was calculated,¹⁰ which is within the range of the general population (6-17%).¹¹ Case series and studies on comorbid OSAHS and epilepsy have reported an improvement in seizure frequency after OSAHS treatment,¹²⁻¹⁵ but the only randomized controlled trial to date¹⁶ did not confirm this observation. The question of above-chance comorbidity of OSAHS and epilepsy could have important treatment implications, but has not yet been fully answered.

Dissociative seizures, also known as psychogenic nonepileptic seizures, are paroxysmal attacks of altered awareness and impaired behavioural control.¹⁷ They can be conceptualized as episodes of acute dissociation in response to perceived threats or conditioned cues,^{18,19} and

Accepted Article

afflicted patients have high rates of dissociative experiences and dissociative disorders.^{17,18} Interestingly, patients frequently report symptoms of disordered sleep such as insomnia, fragmented sleep, daytime sleepiness and fatigue.²⁰ A recent study showed that patients with dissociative seizures more frequently reported moderate to severe changes in sleep patterns than patients with epilepsy, and those changes were associated with worse quality of life.²¹ However, sleep and sleep disorders have rarely been the focus of studies on dissociative seizures.²² Only one small study on 8 women with dissociative seizures has so far systematically investigated sleep quality in this disorder, suggesting a higher ratio of rapid eye movement (REM) sleep compared to 10 epilepsy patients.²³ Most other studies are case reports or case series that examine nocturnal dissociative seizures.²⁴⁻²⁸

A systematic exploratory study of sleep in unselected patients with seizure disorders referred to video-telemetry might elucidate questions of comorbidity, pathophysiological connections and therapeutic leverage. The aim of this study was therefore to identify the prevalence of sleep disorders, particularly OSAHS, sleep-disordered breathing (SDB) and periodic limb movement disorder, and measure objective sleep quality, in a prospectively recruited population with seizure disorders during a diagnostic video EEG monitoring inpatient stay.

METHODS

Participants

All patients of at least 18 years of age due for admission for a five-day clinical video EEG monitoring study at the Western General Hospital in Edinburgh between September 2014 and September 2016, were invited to enter the study either at the time the recommendation for monitoring was made or on arrival to the ward. Informed consent was obtained from all

participants prior to inclusion. This study received ethics approval by the Institutional Review Board (NHS Lothian HREC).

Procedures

Clinical characteristics were recorded as follows: seizure diagnosis (dissociative seizures or epilepsy) based on the results of video EEG monitoring was derived from discharge letters; sleep disorders were diagnosed based on the polysomnography (PSG) results (see below); additional demographic and clinical data such as medication on night of PSG was extracted from electronic medical records. On examination the following anatomical measurements were obtained: weight and height to calculate body mass index (BMI), upper airway shape (assessed with Malampatti classification) and jaw occlusion (using Malocclusion classification). The Epworth sleepiness scale (ESS) was used to assess daytime sleepiness and the generally accepted cut-off of > 10 points (max. 24 points) was used.²⁹

Video PSG was performed on the third night of the monitoring period by adding necessary electrodes and sensors to the video-EEG monitoring setup according to the guidelines from the American Academy of Sleep Medicine. PSG data was scored by a sleep disorders specialist and certified scorer according to above mentioned guidelines without knowledge of the final seizure diagnosis. The following parameters were recorded or derived: sleep latency; total sleep time (TST); time awake after sleep onset in minutes (WASO); time awake after sleep onset as proportion of total duration of sleep ($WASO\% = WASO/(WASO+TST)$); durations of rapid eye movement (REM) sleep and non-REM sleep stages (N1, N2 and N3) in minutes and as percentage of TST; and arousal index (AI). The apnea-hypopnea index (AHI) was calculated as the average number of apnea and hypopnea events per hour of sleep. Periodic limb movement (PLM) was indexed as PLM per hour.

Obstructive sleep apnea-hypopnea syndrome

OSAHS is a disorder of repeated upper airway obstruction with cessation or reduction of airflow (apnea/hypopnea) leading to oxygen desaturation in the blood and arousals. The resultant sleep fragmentation is associated with symptoms of excessive daytime sleepiness.¹¹ The diagnosis of OSAHS depends on the method of examination and varies in the sleep disorders literature.¹¹ Many studies use only polysomnographic indices (such as AI or AHI) to determine the presence of SDB or OSAHS.¹¹ According to the International Classification of Sleep Disorders, 3rd edition, OSAHS diagnosis requires either typical subjective symptoms (significant daytime sleepiness, fatigue, insomnia, snoring, subjective nocturnal respiratory disturbance, or observed apnea) and five or more obstructive respiratory events per hour of sleep; or 15 or more obstructive respiratory events per hour of sleep regardless of reported symptoms.³⁰ Only the ESS was used to assess subjective sleep-related symptoms in this study. Thus, OSAHS was diagnosed when ESS was abnormal (>10) and AHI was between 5 and 15 (mild), or when AHI was between 15 and 30 (moderate) or higher than 30 (severe) regardless of ESS score. Cases in which AHI was between 5 and 10 but the ESS was normal (≤ 10) were tagged as mild SDB.

Periodic limb movements in sleep

Periodic limb movement (PLM), usually of the legs, can occur during sleep and can be associated with physiological arousal and potentially sleep fragmentation.^{31,32} Quantified as movements/hour, a PLM index $> 15/h$ is usually considered abnormal and, in combination with other typical symptoms, supportive of restless legs syndrome. However, in a population-based study of 2162 subjects the prevalence of a PLM index > 15 was 28.6%.³² Periodic limb movement disorder is usually diagnosed when PLM occur during sleep with a PLM index $> 15/h$, the patient reports sleep disturbance or daytime sleepiness, and there is no other

underlying cause such as restless legs syndrome or certain medications.³⁰ Accordingly, we classified patients as “probable PLM disorder” when they fulfilled the following criteria: PLM index >15/h; no concomitant OSAHS diagnosis; either a pathological ESS (ESS>10) or abnormally high arousal index (AI>10).

RESULTS

During the study period a total of 82 patients were considered for participation in the study. Overall, 66 patients were recruited and completed the study. Of these, 22 were found to have dissociative seizures, and 44 had epileptic seizures. The groups did not differ significantly in age, gender distribution or BMI. Malocclusion and Mallampati distribution was comparable and unremarkable. Both groups had relatively high rates of centrally acting medication on the night of PSG. Table 1 presents a summary of demographic, anatomical and clinical characteristics.

Table 2 offers a summary of PSG data comparing both patient groups. There were no significant between-group differences in ESS, AHI, sleep latency, WASO, WASO%, the duration of sleep stages N1, N2 and REM, PLM index, and AI. The average proportion of N3 was significantly higher in the epilepsy group than the dissociative seizures group (30% vs 20.4%, $p=0.008$).

In the epilepsy group ($n = 44$), five patients had an AHI between 5 and 15, but only one of these had an abnormal ESS score fulfilling our criteria for mild OSAHS, and the other four were classified as mild SDB. Two patients had $AHI \geq 15$ (and <30) with normal ESS (≤ 10), fulfilling our criteria for moderate OSAHS. The average BMI of the 4 SDB patients was 26.8 kg/m², with 3 of them in the “overweight” range ($BMI > 25$ kg/m²). The three patients with

OSAHS had an average BMI of 26,1 kg/m². No epilepsy patient fulfilled our criteria for severe OSAHS.

Of the 22 patients with dissociative seizures, two patients (9%) had an AHI between 5 and 15 and ESS>10 (mild OSAHS), and one patient (5%) had moderate OSAHS according to our criteria (AHI: 20.4). The average BMI of these three patients was 38.0 kg/m², with all of them in the “obese” range (BMI > 30 kg/m²). Two patients had mild SDB, one of them “obese” (BMI = 35.5 kg/m²), the other “overweight” (BMI = 25.5 kg/m²). No patient with dissociative seizures fulfilled our criteria for severe OSAHS.

In the epilepsy group, five patients (11%) had a PLM index over 15/h, and 4 (9%) of those fulfilled our criteria for probable PLM disorder. In the dissociative seizures group, seven patients (32%) had a PLM index > 15/h and six (27%) classified as probable PLM disorder.

DISCUSSION

In 44 prospectively recruited epilepsy patients, video PSG revealed only three cases of mild or moderate OSAHS (7%) and 4 cases of SDB (9%). Most of these patients were overweight or obese. In 22 patients with dissociative seizures, 2 (9%) had mild SDB, 2 (9%) had mild OSAHS and one (5%) had moderate OSAHS. All but one of them were obese. These rates of sleep disturbance are in line with rates reported for the general population¹¹ and are in contrast with previous similarly powered studies investigating OSAHS in refractory epilepsy.⁷⁻⁹ This might be explained in part by a difference in OSAHS definitions used. For example, in the oft-cited study by Malow and colleagues obstructive sleep apnea (OSA) diagnosis was established only using the respiratory disturbance index, without taking into account subjective symptoms.⁷ In fact, while OSA was determined in one third of patients, there was no significant difference in ESS scores between patient groups, with the OSA-group having

an average ESS within the normal range. In our study, most patients with SDB or OSAHS were overweight or obese, suggesting that sleep pathology might be more directly related to obesity rather than the seizure disorder.

Concerning PLM, the PLM indices of our patients have to be considered in the context of epidemiological studies of the general population, which have shown rates of PLM > 15/h in 28.6% of adults.³² Although our study was not designed to detect PLM disorder with clinical accuracy or confidence, using AI and ESS as proxies for relevant sleep disturbance, we found a surprising rate of probable PLM disorder in patients with dissociative seizures (27%, compared to 9% in the epilepsy group). On a related note, WASO% was relatively high in both groups (25.7% and 22.7% respectively) with patients from both groups spending an average of 2 hours awake each night, which is very high compared to healthy adults.³³

High WASO rates are a marker of sleep disruption. This can result in relative sleep deprivation, a well-recognized precipitant for epileptiform discharges and seizures.³ Sleep deprivation has been shown to lead to an increase in cortical excitability,³⁴ which is even more pronounced in patients with idiopathic generalized epilepsy.³⁵ In cases when sleep deprivation (or sleep inefficiency) is due to OSAHS, respective treatment might indeed lead to a reduction in seizure frequency as discussed above. However, our study does not support the claim that OSAHS (or SDB) is a particularly common cause of sleep disruption in epilepsy patients. If a patient with epilepsy reports sleep disturbances, other factors such as medication effects, psychiatric comorbidities and nocturnal epileptic activity must all be considered; this study suggests these are potentially more likely than OSAHS to be responsible.

The high WASO and PLM rates in dissociative seizures patients might be more than incidental findings. Studies have suggested a causal association between disordered sleep and

dissociative experiences.³⁶ Acute sleep loss of 24 or 36 hours in an experimental setting can enhance dissociative experiences in healthy controls as assessed by self-report scales and cognitive tasks.^{37,38} Clinically insomniac patients score high on the Dissociative Experiences Scale with scores correlated to quantifiable EEG parameters.³⁹ Conversely, in a mixed psychiatric inpatient sample an improvement in sleep quality led to a decrease in dissociative experiences.⁴⁰ Our polysomnographic findings in patients with dissociative seizures suggest that sleep deprivation might be a common occurrence in these patients, supporting a possible contribution to the propensity for dissociative attacks. Further studies are needed to elucidate this relationship, since in some cases it might reveal relevant treatment opportunities.

Our study has several important limitations. Only the ESS was used to assess relevant symptoms of OSAHS, potentially missing the diagnosis of mild OSASH in individuals with mild SDB (AHI between 5 and 15) and other diagnostically relevant symptoms such as insomnia or snoring. Video PSG is usually performed in dedicated sleep laboratories, where special precautions are taken to ensure a sufficient level of comfort for patients, which is not achieved in quite the same level in an epilepsy monitoring unit. Many of our patients were under the influence of centrally acting medication, which are known to affect sleep through various mechanisms. However, while possibly obscuring pathophysiological effects between sleep and seizure disorders, this reflects the reality of clinical care in these patients. Due to its exploratory nature, the study was not powered strongly enough to clarify all reported findings and avoid possible type I errors. Also, the lack of healthy controls weakens the validity of some judgements beyond simple between-group analyses. Patients were selected based on the recommendation of telemetry (and patient consent), so there is a selection bias which is difficult to define and limits generalisability. Finally, PSG was performed one night only. However, it was performed on the third night of admission to reduce possible “first night”

Accepted Article

effects (although studies have suggested that any such effects on PSG parameters may be minor⁴¹).

In conclusion, our findings do not support claims of high comorbidity of OSAHS and epilepsy; raise the suspicion of high rates of periodic limb movement disorder in patients with dissociative seizures; and reveal high WASO times in both epilepsy and dissociative seizures patients. While the relevance of sleep deprivation for epilepsy is well known, its role in the pathogenesis of dissociative seizures is unknown. Considering its high prevalence, further studies on sleep and dissociative seizures are warranted.

ACKNOWLEDGMENTS

The authors would like to thank Stevie Williams for polysomnography data acquisition.

DISCLOSURE OF POTENTIAL CONFLICTS OF INTEREST

None.

REFERENCES

1. Jackson JH. Lectures on the Diagnosis of Epilepsy. *British Medical Journal* 1879; **1**: 141–143.
2. Janet P. The major symptoms of hysteria. London: Macmillan; 1907.
3. Derry CP, Duncan S. Sleep and epilepsy. *Epilepsy & Behavior* 2013; **26**: 394–404.
4. Ali A, Wu S, Issa NP, et al. Association of sleep with sudden unexpected death in epilepsy. *Epilepsy & Behavior* 2017; **76**: 1–6.
5. Bazil CW. Sleep and Epilepsy. *Seminars in Neurology* 2017; **37**: 407–412.

6. Lin Z, Si Q, Xiaoyi Z. Obstructive sleep apnoea in patients with epilepsy: a meta-analysis. *Sleep and Breathing* 2017; **21**: 263–270.
7. Malow BA, Levy K, Maturen K, Bowes R. Obstructive sleep apnea is common in medically refractory epilepsy patients. *Neurology* 2000; **55**: 1002–1007.
8. Malow BA, Weatherwax KJ, Chervin RD, et al. Identification and treatment of obstructive sleep apnea in adults and children with epilepsy: a prospective pilot study. *Sleep Medicine* 2003; **4**: 509–515.
9. Weatherwax KJ, Lin X, Marzec ML, Malow BA. Obstructive sleep apnea in epilepsy patients: the Sleep Apnea scale of the Sleep Disorders Questionnaire (SA-SDQ) is a useful screening instrument for obstructive sleep apnea in a disease-specific population. *Sleep Medicine* 2003; **4**: 517–521.
10. Manni R, Terzaghi M, Arbasino C, Sartori I, Galimberti CA, Tartara A. Obstructive sleep apnea in a clinical series of adult epilepsy patients: frequency and features of the comorbidity. *Epilepsia* 2003; **44**: 836–840.
11. Senaratna CV, Perret JL, Lodge CJ, et al. Prevalence of obstructive sleep apnea in the general population: A systematic review. *Sleep Medicine Reviews* 2017; **34**: 70–81.
12. Devinsky O, Ehrenberg B, Barthlen GM, Abramson HS, Luciano D. Epilepsy and sleep apnea syndrome. *Neurology* 1994; **44**: 2060–2064.
13. Hollinger P, Khatami R, Gugger M, Hess CW, Bassetti CL. Epilepsy and obstructive sleep apnea. *European Neurology* 2006; **55**: 74–79.
14. Vaughn BV, D'Cruz OF, Beach R, Messenheimer JA. Improvement of epileptic seizure control with treatment of obstructive sleep apnoea. *Seizure* 1996; **5**: 73–78.
15. Vendrame M, Auerbach S, Loddenkemper T, Kothare S, Montouris G. Effect of continuous positive airway pressure treatment on seizure control in patients with obstructive sleep apnea and epilepsy. *Epilepsia* 2011; **52**: e168–e171.

- Accepted Article
16. Malow BA, Foldvary-Schaefer N, Vaughn BV, et al. Treating obstructive sleep apnea in adults with epilepsy: a randomized pilot trial. *Neurology* 2008; **71**: 572–577.
 17. Brown RJ, Reuber M. Psychological and psychiatric aspects of psychogenic non-epileptic seizures (PNES): A systematic review. *Clinical Psychology Review* 2016; **45**: 157–182.
 18. Bowman ES. Why conversion seizures should be classified as a dissociative disorder. *Psychiatric Clinics of North America* 2006; **29**: 185–211.
 19. Stone J, Carson AJ. The unbearable lightheadedness of seizing: wilful submission to dissociative (non-epileptic) seizures. *Journal of Neurology, Neurosurgery, and Psychiatry* 2013; **84**: 822–824.
 20. Graham CD, Kyle SD. A preliminary investigation of sleep quality in functional neurological disorders: Poor sleep appears common, and is associated with functional impairment. *Journal of Neurological Sciences* 2017;378:163-6.
 21. Latreille V, Baslet G, Sarkis R, Pavlova M, Dworetzky BA. Sleep in psychogenic nonepileptic seizures: Time to raise a red flag. *Epilepsy & Behavior*. 2018; **86**: 6–8.
 22. Pavlova MK, Allen RM, Dworetzky BA. Sleep in psychogenic nonepileptic seizures and related disorders. *Clinical EEG and Neuroscience* 2015; **46**: 34–41.
 23. Bazil CW, Legros B, Kenny E. Sleep structure in patients with psychogenic nonepileptic seizures. *Epilepsy & Behavior* 2003; **4**: 395–398.
 24. Benbadis SR, O'Neill E, Tatum WO, Heriaud L. Outcome of prolonged video-EEG monitoring at a typical referral epilepsy center. *Epilepsia* 2004; **45**: 1150–1153.
 25. Duncan R, Oto M, Russell AJ, Conway P. Pseudosleep events in patients with psychogenic non-epileptic seizures: prevalence and associations. *Journal of Neurology, Neurosurgery, and Psychiatry* 2004; **75**: 1009–1012.
 26. Thacker K, Devinsky O, Perrine K, Alper K, Luciano D. Nonepileptic seizures during apparent sleep. *Annals of Neurology* 1993; **33**: 414–418.

27. Orbach D, Ritaccio A, Devinsky O. Psychogenic, nonepileptic seizures associated with video-EEG-verified sleep. *Epilepsia* 2003; **44**: 64–68.
28. Seneviratne U, Minato E, Paul E. Seizures by the clock: Temporal patterns of psychogenic nonepileptic seizures. *Epilepsy & Behavior* 2017; **76**: 71–75.
29. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991; **14**: 540–545.
30. American Academy of Sleep Medicine. International Classification of Sleep Disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
31. Frauscher B, Gabelia D, Mitterling T, et al. Motor events during healthy sleep: a quantitative polysomnographic study. *Sleep* 2014; **37**: 763–773, 773A–773B.
32. Haba-Rubio J, Marti-Soler H, Marques-Vidal P, et al. Prevalence and determinants of periodic limb movements in the general population. *Annals of Neurology* 2016; **79**: 464–474.
33. Ohayon MM, Carskadon MA, Guilleminault C, Vitiello MV. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. *Sleep* 2004; **27**: 1255–1273.
34. Huber R, Mäki H, Rosanova M, et al. Human cortical excitability increases with time awake. *Cerebral Cortex* 2013; **23**: 332–338.
35. Badawy RA, Curatolo JM, Newton M, Berkovic SF, Macdonell RA. Sleep deprivation increases cortical excitability in epilepsy: syndrome-specific effects. *Neurology* 2006; **67**: 1018–1022.
36. van der Kloet D, Merckelbach H, Giesbrecht T, Lynn SJ. Fragmented Sleep, Fragmented Mind: The Role of Sleep in Dissociative Symptoms. *Perspectives on Psychological Science* 2012; **7**: 159–175.

- Accepted Article
37. Giesbrecht T, Smeets T, Leppink J, Jellic M, Merckelbach H. Acute dissociation after 1 night of sleep loss. *Journal of Abnormal Psychology* 2007; **116**: 599–606.
 38. van Heugten-van der Kloet D, Giesbrecht T, Merckelbach H. Sleep loss increases dissociation and affects memory for emotional stimuli. *Journal of Behavior Therapy and Experimental Psychiatry* 2015; **47**: 9–17.
 39. Van Der Kloet D, Giesbrecht T, Franck E, et al. Dissociative symptoms and sleep parameters--an all-night polysomnography study in patients with insomnia. *Comprehensive Psychiatry* 2013; **54**: 658–664.
 40. van der Kloet D, Giesbrecht T, Lynn SJ, Merckelbach H, de Zutter A. Sleep normalization and decrease in dissociative experiences: evaluation in an inpatient sample. *Journal of Abnormal Psychology* 2012; **121**: 140–150.
 41. Selwa LM, Marzec ML, Chervin RD, et al. Sleep staging and respiratory events in refractory epilepsy patients: Is there a first night effect? *Epilepsia* 2008; **49**: 2063–2068.

Table 1: Demographic, anatomical and clinical characteristics.

Characteristics	Dissociative seizures	Epilepsy	<i>ttest p</i>
Group size	n = 22	n = 44	
Age, mean (SD)	41.8 (12.7)	38.4 (10.7)	0.29
Female sex	14 (64%)	24 (55%)	
Body mass index, mean (SD)	27.9 (7.0)	27.1 (4.8)	0.64
% overweight (25-30)	5 (23%)	16 (36%)	
% obese (>30)	5 (23%)	13 (30%)	
Mallampati			
Class 1	10 (45%)	11 (26%)	
Class 2	10 (45%)	27 (64%)	
Class 3	2 (9%)	3 (7%)	
Class 4	0 (0%)	1 (2%)	
Malocclusion			
Normal	16 (73%)	31 (74%)	
Class 1	3 (14%)	3 (7%)	
Class 2	3 (14%)	7 (17%)	
Class 3	0 (0%)	1 (2%)	
Medication on night of PSG			
Benzodiazepines	2 (11%)	3 (8%)	
Z-drugs	0	0	
Opiates	3 (16%)	4 (10%)	
Antidepressants	4 (21%)	6 (15%)	
Antiepileptic drugs	8 (42%)	35 (88%)	

Data presented as mean (SD) or number (percent of group sample) unless noted otherwise.

Table 2: Sleep questionnaire and polysomnography parameters

	Dissociative seizures	Epilepsy	<i>ttest p</i>
Group size	n = 22*	n = 44*	
Sleep latency, min	41.5 (40.1)	37.9 (47.1)	0.76
TST, min	365.3 (80.8)	379.5 (107.2)	0.56
WASO,min	124.6 (85.4)	110.4 (95.6)	0.55
WASO %, %	25.7 (14.8)	22.7 (19.6)	0.50
N1, min	16.4 (13.1)	14.3 (10.4)	0.51
N1 as % of TST	4.7 (4.1)	4.0 (3.7)	0.53
N2, min	202.2 (73.2)	218.2 (218.7)	0.67
N2 as % of TST	55.5 (16.2)	49.3 (12.1)	0.13
N3, min	75.5 (43.1)	106.8 (46.8)	0.011
N3 as % of TST	20.4 (11.7)	30.0 (15.7)	0.008
REM, min	64.0 (27.5)	66.6 (36.3)	0.75
REM as % of TST	18.1 (7.8)	16.7 (8.0)	0.52
ESS, mean	8.3 (4.9)	7.5 (4.5)	0.55
> 10 points, n (%)	8 (36%)	13 (31%)	
AHI, mean/h	3.5 (4.8)	2.8 (4.1)	0.60
AHI<5, n (%)	17 (77%)	37 (84%)	
PLM index (PLM/h), mean	13.8 (19.6)	7.4 (16.0)	0.20
PLM index > 15/h, n (%)	7 (32%)	5 (11%)	
AI, arousals/h	8.2 (5.3)	6.0 (4.4)	0.11
SDB			
Mild SDB (ESS≤10 + 5≤AHI<15), n (%)	2	4 (9%)	
OSAHS			
Mild OSAHS (ESS>10 + AHI≥5, but <15), n (%)	2 (9%)	1 (2%)	
Moderate OSAHS (15≤AHI<30), n (%)	1 (5%)	2 (5%)	
Severe OSAHS (AHI≥30), n (%)	0	0	
Probable PLM disorder ([PLM index > 15 + AI>10/h] or [PLM index > 15 + ESS>10])	6 (27%)	4 (9%)	

Data presented as mean (SD) or number (percent of group sample) unless noted otherwise.

*for technical reasons some datasets were incomplete: 2 epilepsy patients missing ESS scores; 3 dissociative seizures patients and 4 epilepsy patients missing information on medication on night of study; 1 dissociative seizures patient and 4 epilepsy patients did not have AI data.

Abbreviations: AI, arousal index; AHI, apnea hypopnea index; Epworth sleepiness scale; OSAHS, obstructive sleep apnea-hypopnea syndrome; PLM, periodic limb movement; REM, rapid eye movement; SDB, sleep-disordered breathing; TST, total sleep time; WASO, wake after sleep onset.